cerebral hemorrhage could with probability be diagnosed. I have described a case of acute leukemia mistaken for cerebrospinal meningitis, the clinical findings of which pointed to a leukemic infiltration as the cause of the symptoms. A very similar case with autopsy is reported from the literature. Finally, I have described a case with terminal cerebral symptoms in which areas of macroscopic leukemic infiltration were found at postmortem examination.

I wish to express my indebtedness to Dr. Henry Heiman for permission to make use of the material from the pediatric service of Mount Sinai Hospital.

PERICARDITIS: INCIDENCE AND DIAGNOSIS.[1]

By W. W. G. MacLachlan, M.D.,
ASSISTANT PROFESSOR OF MEDICINE, UNIVERSITY OF PITTSBURGH, PITTSBURGH, PA.

Inflammation is prone to develop in the serous sacs of the body, in some, seemingly, more than in others. The inflammatory reactions demonstrate nicely the various types of inflammations; in fact, in no situation in the body is one able to observe a more distinct set of reactions than are met with in the different forms of serositis. With the exception of direct wounds, infection in serous sacs is always a secondary manifestation of some primary focus, even though, at times, this focus may be so completely hidden as to escape recognition. One usually does not regard epidemic meningitis exactly in this light, but really the invasion from the upper respiratory tract of the meningococcus with a subsequent meningitis or of the Streptococcus salivarius with its accompanying arthritis or pericarditis is theoretically the same. There is no reason to believe that serous sacs are not infected in the usual ways—namely, by the blood stream, the lymphatics or by direct extension. It is, however, impossible in some instances of pericarditis to be certain of the manner of infection, hence the term idiopathic has been applied to certain clinical forms of the disease. On general medical grounds this term is a poor one, and even though, from the point of view of proof, it is at times correct, still it is probably better to drop the word idiopathic. The pericardium is subject to four types of acute inflammation; the acute fibrinous and the serofibrinous are the common forms while the purulent and the hemorrhagic forms are less frequent. The chronic inflammations are indicated by fibrous adhesions, localized or general in distribution, and a rather unusual form in which a diffuse thickening of the pericardium is observed. Tuberculosis produces an inflammatory reaction which at times shows characteristics typical of both acute and chronic inflammation in addition to the granulomatous reaction.

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[1] Read at the Interurban Clinical Society, Clifton Springs Sanatorium, October, 1920.

It is quite apparent from clinical and pathological observations that the different serous sacs are by no means numerically equally involved in infection. There is no reason to believe that any inherent variability in the power of the resistance is possessed by one and not by the other. It would seem, rather, that the anatomical position in the body on the one hand and the type of bacterial infection on the other suggests the explanation. For example the acute inflammations of the peritoneum are practically always purulent, because they are secondary in most instances to a ruptured or leaking hollow viscus containing pus-producing bacteria. The pericardium, however, has choicer surroundings as the rupture of a viscus into it is extremely unusual; while, further, the Streptococcus pyogenes is not the prevailing organism in inflammations of the chest, the possible regional source of pericarditis. On the other hand the frequency of pericarditis in the Streptococcus viridans infection and the infrequency of any peritoneal involvement by this organism, at least that can be recognized as such, would appear to indicate that the type of bacterium is a factor of importance in inflammations of serous membranes. In the same way, Streptococcus viridans infection of the meninges, except in an occasional case of chorea, is very unusual; but it undoubtedly occurs in the pleura. It is not our purpose to become entangled in the bacteriological question of specific localization of bacteria which, in a certain sense, is correct as the localization of the meningococcus in the meninges, the tendency for Streptococcus viridans to be present in carditis, while the absence of these bacteria in the peritoneum certainly would suggest specificity in location. This, however, under no condition means that Streptococcus viridans isolated from a pericardial sac will always produce a pericarditis. Holman, McMeans and others have settled this point. The prevalence, therefore, of acute fibrinous or serofibrinous inflammations in the pericardium would appear to be due, firstly, to the fact that the most frequent bacterial invader is the Streptococcus viridans, and, secondly, the pericardium is not especially subjected to the influence of the usual pus-producer, the Streptococcus pyogenes. That the pericardium is readily available for a purulent infection if the pus-producing organism is present, is clearly shown by the considerable number of purulent pericarditis cases found in streptococcic pneumonias and to a lesser extent in the pneumonia of pneumococcic origin.

It is my purpose to deal with the pericardium in inflammatory conditions as recognized at the bedside and at the autopsy table. It must be kept in mind that acute pericarditis, with or without effusion, has not a high mortality and therefore the cases that come to autopsy, dying from acute pericarditis *per se*, are unusual. Further, much of the acute pericarditis found at autopsy is but a terminal part of the picture of infection in that individual. Chronic pericarditis is of interest in indicating the number of cured acute forms and also in calling attention to clinical difficulties of diagnosis in this condition, for it still seems to be a fact, as Osler remarked many years ago, that chronic pericarditis is more often found in the autopsy room than in the ward. I thought it would be of some interest to review our autopsy records on this subject and to collect from them any data which seemed to have a pertinent bearing on the subject of pericarditis.

The subject of pericarditis is an old one, and in the literature it has been thoroughly covered as the reports, which are legion, on this disease would indicate. Our pathological findings are in accord with what has been shown in the past, our figures bearing a striking resemblance to those published about twenty years ago by Wells. One is also forced to say there is little further to add to the diagnosis; but as we are still going to have pericarditis with us it is possibly not out of place to occasionally submit a review. The number of autopsies examined was 975; in 100 of these pericarditis was present in some form. For the opportunity to study these records and for the helpful discussion with reference to them I wish to thank Dr. Oskar Klotz.

There are two conditions to which brief reference should be made, although neither of them is classed under inflammation. Hydropericardium has clinical significance in that some degree of this transudation is present when edema is a characteristic of certain chronic diseases, especially nephritis and primary anemias. A well-marked increase of fluid in the pericardial sac without other signs of pericarditis, or the presence in the body of edema in the tissues or fluid in the cavities, should be held in suspicion as probable evidence of the inflammation, the reaction being principally of a serous character. This differentiation, however, does not appear to be a difficult problem to decide, either from the clinical or the pathological point of view. A milk spot on the surface of the heart is, to say the least, a rather common finding. About the origin of these spots there is considerable debate, as the question has been raised as to whether they represent the end-results of inflammation or merely the effects of long-continued rubbing of the pericardial surfaces. It is not my intention to attempt to settle this problem in one way or the other, notwithstanding the fact that I have observed at least some milk spots to be of an inflammatory nature. They occur at all ages. Our records show 2 examples in infants, but they are undoubtedly more common past the middle period of life. It seems rather strange that we should not know exactly why these plaques develop as they are certainly accessible to study.

Under this heading we have the acute fibrinous, serofibrinous and purulent forms of pericarditis. The first two types predominate; gradations in the purulent forms occur from the slightly turbid fluid to a frankly purulent one. The latter type has been unusually infrequent in our series of cases. There are several points brought out in the table which would bear some analysis. It is at once seen that a little over 5 per cent of the autopsies showed an acute pericarditis, and of this number the males were more than three times as numerous as the females, bearing out the general known sex incidence of this disease. Rather strangely, however, more cases occur after the age of forty. This is likely due to two facts, the number of children in our series was relatively small and acute pericarditis, *per se*, is not often fatal. It should be kept in mind that there is a difference in the group of acute pericarditis cases met with in the wards and in the autopsy room, especially in regard to the age. As a clinical disease, acute pericarditis is more often observed in young adults and children; further, it has a relatively low mortality, and hence is not frequently seen at autopsy. On the other hand, there are many instances of acute pericarditis appearing as a terminal infection with some other acute disease which was the cause of death. The involvement of the pericardium in this type of case is frequently not recognized before death; while it may be present at any age, our results indicated the tendency of this form to develop more often after middle life.

TABLE I.—ACUTE PERICARDITIS.

Cases.	Sex.		Age.		Amount of fluid.	
50	Males	39	up to 10	2	5 c.c.	1
	Females	11	10 to 20	6	10 c.c.	1
			20 to 30	7	15 c.c.	1
			30 to 40	9	20 c.c.	2
			40 to 50	10	50 c.c.	12
			50 to 60	7	60 c.c.	1
			60 to 70	8	70 c.c.	1
			70 to 80	1	100 c.c.	5
					150 c.c.	2
					200 c.c.	2
					300 c.c.	1
					400 c.c.	1
					600 c.c.	1
					Stated as not increased	19

Acute pericarditis does not necessarily produce an increase in the amount of fluid in the pericardial sac. Almost three-fourths of our cases showed 50 cc or less fluid in the sac, while but one-seventh of the number had more than 100 cc of fluid present. This would seem to indicate, from the autopsy point of view, that the majority of acute infections in the pericardium are not associated with a large amount of fluid; but, as we mentioned previously, it is well to keep in mind that the acute serofibrinous pericarditis seen in the younger individual rarely comes to the autopsy table by reason of that disease alone. The reliance on increased cardiac dulness, assuming that it is due to fluid, as a point of diagnostic value may